A COMPARATIVE STUDY ON THE EFFICACY OF MAGNESIUM SULPHATE WITH LIGNOCAINE IN ATTENUATING THE CARDIOVASCULAR RESPONSES TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

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BRANCH – X (Anaesthesiology)

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CHENNAI

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CERTIFICATE

This is to certify that this dissertation entitled

"A COMPARATIVE STUDY ON THE EFFICACY OF MAGNESIUM SULPHATE WITH LIGNOCAINE IN ATTENUATING THE CARDIOVASCULAR RESPONSES TO LARYNGOSCOPY AND INTUBATION"

is a bonafide record of the work done by DR. S. NARMATHA YANGTSE under my supervision and guidance in the Department of Anesthesiology at Thanjavur Medical College Hospital, Thanjavur, during the period of her postgraduate study from May 2006 to March 2009 for the partial fulfillment of M.D. (Branch – X Anesthesiology) degree.

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Last but not the least I am grateful to all my patients who made this study possible.

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INTRODUCTION

INTRODUCTION

Control of airway is one of the defining moments of Anaesthesia.

Before the twentieth century, intubation of the trachea had been described and performed rather crudely, often using fingers as a makeshift laryngoscope without using any pharmacological agents. At that time the only regular intubation of the trachea that was taking place was in the resuscitation of asphyxiated neonate.

In 1880, Sir William Maceven[20], a Scottish surgeon was the first to perform endotracheal intubation.

In 1895 Kirstein became the first to perform endotracheal intubation using a laryngoscope.

The credit of developing the scientific principles of direct laryngoscopy and endotracheal intubation belongs to the American otolaryngologist Dr. Chevallier Jackson. In 1913-jackson devised a U-shaped laryngoscope.

In 1913-Janeway introduced L-shaped laryngoscope with batteries in the handle.

Now we use rigid direct laryngoscopes to view the larynx and adjacent structures under direct vision for the purpose of endotracheal intubation. This causes direct trauma to the oropharynx and larynx and apart from this it also causes sympathetic stimulation resulting in rise in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure.

Although the haemodynamic stress response is transient, and of little consequence in healthy individuals, it is hazardous to those with systemic hypertension, coronary artery disease, and cerebro vascular disease. Complications like myocardial ischaemia, infarction, left ventricular failure, arrhythmias, intracranial hemorrhage can occur due to this response.

The major cause of the haemodynamic stress response is due to the stimulation of supraglottic area by the laryngoscope blade followed by additional stimulation contributed by tracheal tube placement.

Till date the mainstay of attenuation of the haemodynamic stress response was done by using various drugs like local anesthetics, beta-blockers, calcium channel blockers, opiods and vasodilators.

All of these techniques which are suggested have some disadvantages related to

either cardiovascular or respiratory depression, but none of them directly inhibits the release of catecholamines. Magnesium sulphate blocks the release of catecholamines from the adrenergic nerve terminals and adrenal glands.

The role of magnesium starts as back as the 17th century and covers a large span of the chemical and pharmacological fields of knowledge.

Until recently the function of Magnesium in biological processes was largely ignored to the point where it was described as the “forgotten ion”. Magnesium is the fourth most abundant cation in the body and the second abundant intracellular cation. It is involved in several processes like control of vasomotor tone, cardiac excitability and neuro transmitter release. In many of its actions it is likened to a physiological calcium antagonist.

In our study, which was carried out in the Department of Anaesthesiology at Thanjavur medical college hospital we compared intravenous Magnesium sulphate and Lignocaine in the attenuating haemodynamic stress response (increase in heart rate and an increase in the mean arterial pressure) to laryngoscopy and intubation, and find out which drug is better.

AIM

AIM

This study was done with the following intentions:

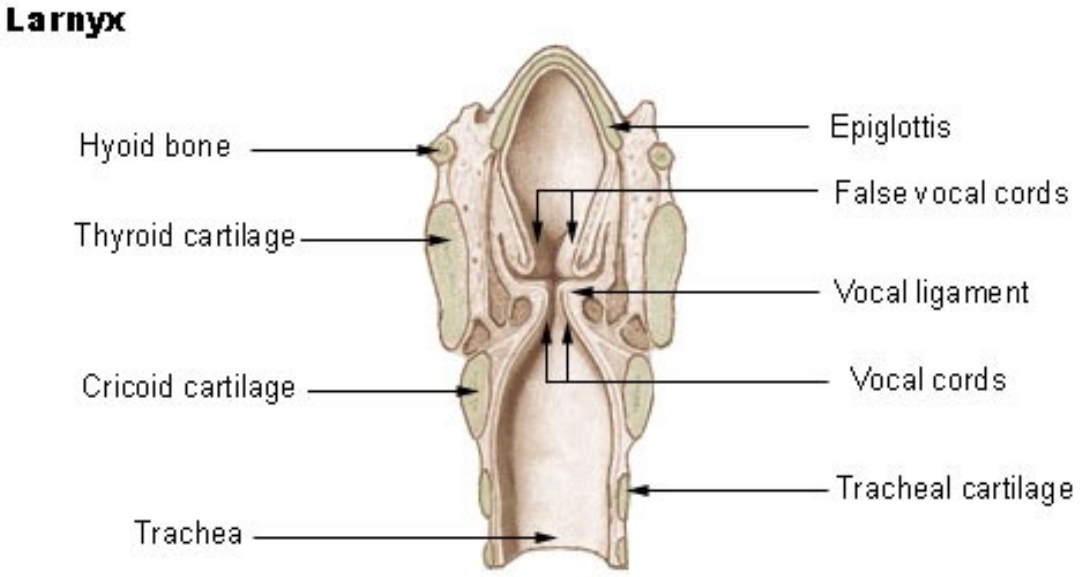
- To compare the efficacy of Magnesium sulphate over Lignocaine in attenuating the cardiovascular responses to
  - Laryngoscopy and
  - Intubation.

- To observe any adverse effects of Magnesium sulphate in the specified dosage.

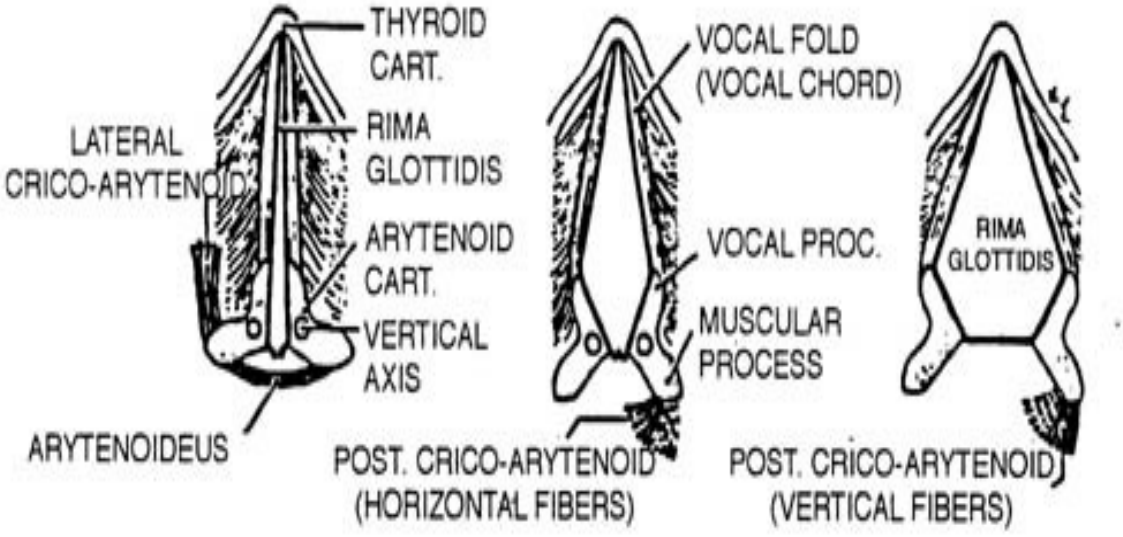
- To observe any prolongation of neuromuscular blockade in this specified dose.

ANATOMY & NERVE SUPPLY OF LARNYX

ANATOMY OF LARYNX



ANATOMY OF VOCAL CORDS



ANATOMY AND NERVE SUPPLY OF THE UPPER AIRWAY[14]

The pharynx is a U shaped fibromuscular structure that extends from the base of the skull to the cricoid cartilage. It opens anteriorly into the nasal cavity, mouth and the larynx, which conveniently divides the pharynx into three parts termed as nasopharynx, oropharynx and laryngopharynx respectively. At the base of the tongue, the epiglottis functionally separate the oropharynx from the laryngopharynx.

Sensory nerve supply of the upper airway is derived from the cranial nerves trigeminal, glossopharyngeal and the vagus.

The palatine nerve provides sensory fibers from trigeminal nerve to hard and soft palate. The lingual nerve provides general sensation to the anterior two thirds of the tongue, and the glossopharyngeal nerve to the posterior one third of the tongue. Branches of the facial nerve and the glossopharyngeal nerve provide sensation of taste to anterior two thirds and posterior one third respectively.

The glossopharyngeal nerve also innervates the roof of the pharynx, the tonsils and the undersurface of the soft palate. The pharyngeal surface of epiglottis is supplied

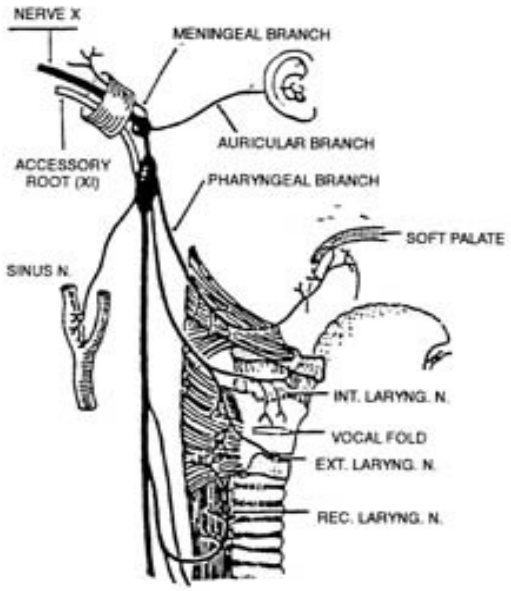
by the glossopharyngeal nerve and the laryngeal surface of the epiglottis is supplied by the vagus nerve. Internal laryngeal branch of superior laryngeal branch provides sensory supply to the supraglottic area. The recurrent laryngeal nerve ascends to the larynx in the groove between the esophagus and trachea and divides into motor and sensory branches.[14]

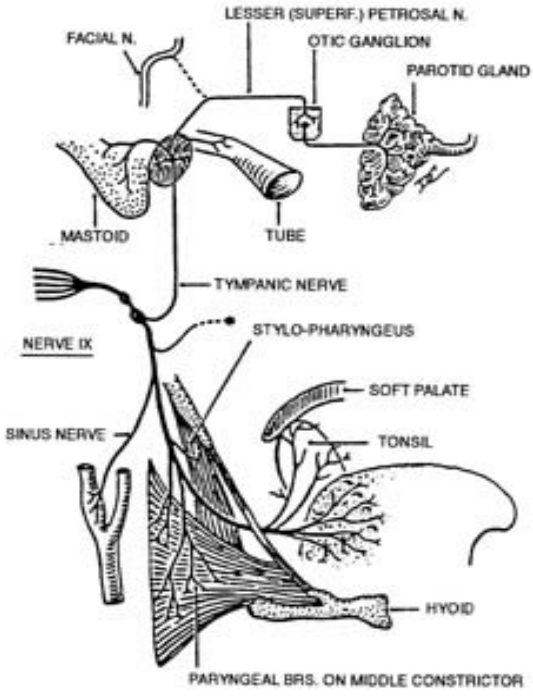
The motor branch supplies all the intrinsic muscles of the larynx except cricothyroid.

The sensory branch supplies the laryngeal mucous membrane below the level of vocal cords.

PHYSIOLOGY OF STRESS RESPONSE

NERVE SUPPLY OF LARYNX





THE VAGUS NERVE AND ITS BRANCHES &
THE GLOSSOPHARNYGEAL NERVE AND ITS BRANCHES

PHYSIOLOGY OF STRESS RESPONSE[17]

Haemodynamic stress response to laryngoscopy and intubation occurs as increase in the heart rate and the mean arterial pressure due to reflex sympathetic discharge in response to laryngo - tracheal stimulation.

Tracheal intubation alters respiratory and cardiovascular physiology by reflex response and also by the physical presence of endotracheal tube. Although these circulatory responses are transient and of little consequence in patients with normal circulatory system, they may be exaggerated in patients with coronary artery disease, reactive airways and intracranial pathology.

CARDIOVASCULAR RESPONSE

This transitory variable and unpredictable response is mediated by both sympathetic and parasympathetic nervous systems. Usually bradycardia seen in neonates and infants during laryngoscopy and intubation is the autonomic equivalent of Laryngospasm response in adults. This reflex is mediated by an increase in vagal tone at the SA node and is virtually a monosynaptic response to a noxious stimuli in the airway.[14, 21]

The more common response to tracheal intubation is hypertension and

tachycardia mediated by sympathetic efferents via the cardioaccelarator nerves and sympathetic chain ganglia .The polysynaptic nature of pathways from the IX and X nerve afferents to the sympathetic nervous system via the brain stem and spinal cord results in a diffuse autonomic response which includes widespread release of norepinephrine from the adrenergic terminals and release of epinephrine from the adrenal medulla.

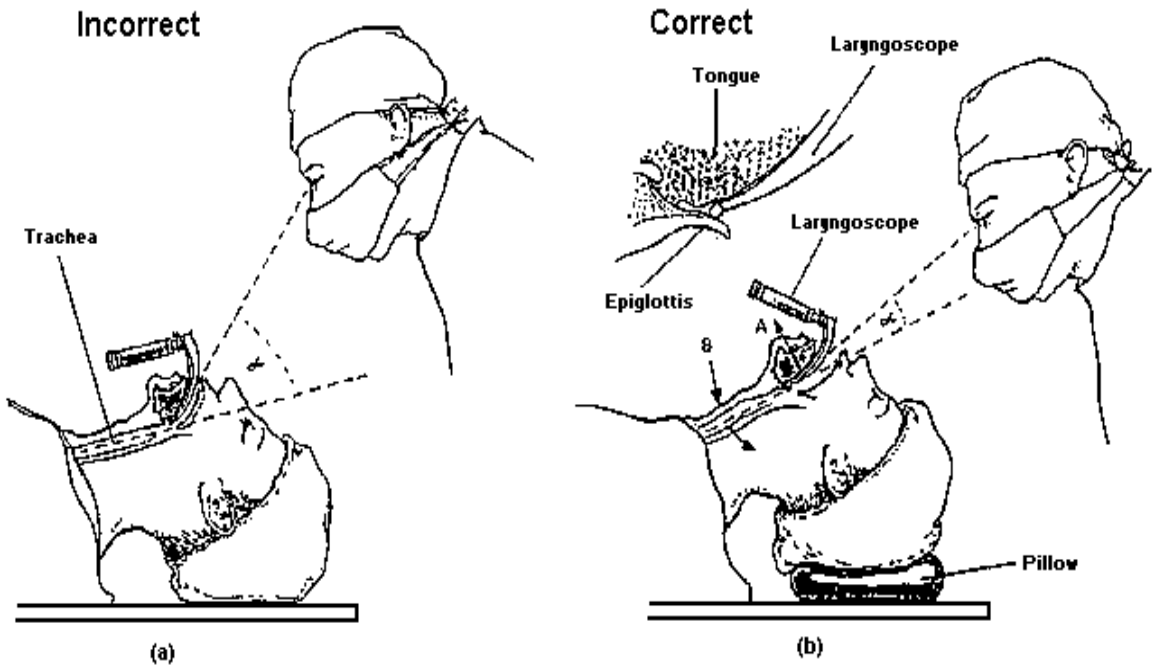
One other reason for the hypertensive response is due to activation of renin-angiotensin system with release of renin from the renal juxtaglomerular apparatus, an end organ innervated by adrenergic nerve terminals.

RESPIRATORY PHYSIOLOGY

- Glottic closure reflex [laryngospasm]
- Reduction in dead space.
- Increase in Airway resistance.
- Bronchospasm as a reflex response to intubation.
- Removes the glottic barrier and may lower lung volume.
- Cough efficiency is reduced.

LARYNGOSCOPY & ENDOTRACHEAL INTUBATION

POSITION FOR ENDOTRACHEAL INTUBATION



LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION[17,20]

Direct laryngoscopy is visualization of the larynx under direct vision by the use of a mechanical device namely laryngoscope.

Endotracheal intubation is the trans laryngeal placement of a tube into the trachea via the nose or mouth.

TECHNIQUE FOR ORO TRACHEAL INTUBATION:

This is a technique which requires training and experience to make it safe, effective and least traumatic.

Equipments needed are laryngoscope, proper size endotracheal tube, appropriate anaesthetic drugs. suction apparatus, multimonitor, and facilities for IPPV of the lungs with oxygen.

Patient is positioned in Modified Jackson or sniffing position, and head is elevated by 10 centimeters with pads under the occiput , the cervical spine is maintained straight and the extension is made at the atlanto occipital joint. So as to make the patient's head in level with the anesthetist's xiphiod cartilage [20]

Procedure:

The laryngoscope is held in the left hand near the junction of the handle and the blade, and is inserted on the right side of the patient's mouth to deflect the tongue away from the line of vision; the laryngoscope handle must be maintained perpendicular to the plane of the patient's body. After placing the blade in the mouth gentleness and avoiding of pressure on the upper teeth or gums are essential.

The handle should never be levered towards the anesthetist and the pull should be in the long axis of the handle. The epiglottis is visualized and the tip of the curved blade is slid into the vallecula between the base of tongue and the epiglottis. The subsequent forward and upward movement of the blade exerted along the axis of the handle stretches the hyoepiglottic ligament causing the epiglottis to move upward and expose the glottic opening [20].

Placement of Endotracheal tube:

The glottic opening is recognized by its triangular shape and the pale white vocal cords. The ETT is held in the hand like a pencil and introduced on the right side of the patient's mouth with the built in curve directed anteriorly. The tube is advanced till the cuff just disappears behind the cords. The laryngoscope is then with drawn and the cuff is inflated after verifying the air entry on both sides.

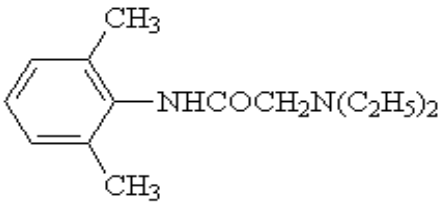
PHARMACOLOGY OF LIGNOCAINE

PHARMACOLOGY OF LIGNOCAINE[8]

Lignocaine was synthesized by Lofgren in Sweden in 1943.

Lofgren and Lundquist discovered the anaesthetic properties of lignocaine in 1948, which was then introduced into clinical practice in 1949 by Gordon.[20]

STRUCTURE:

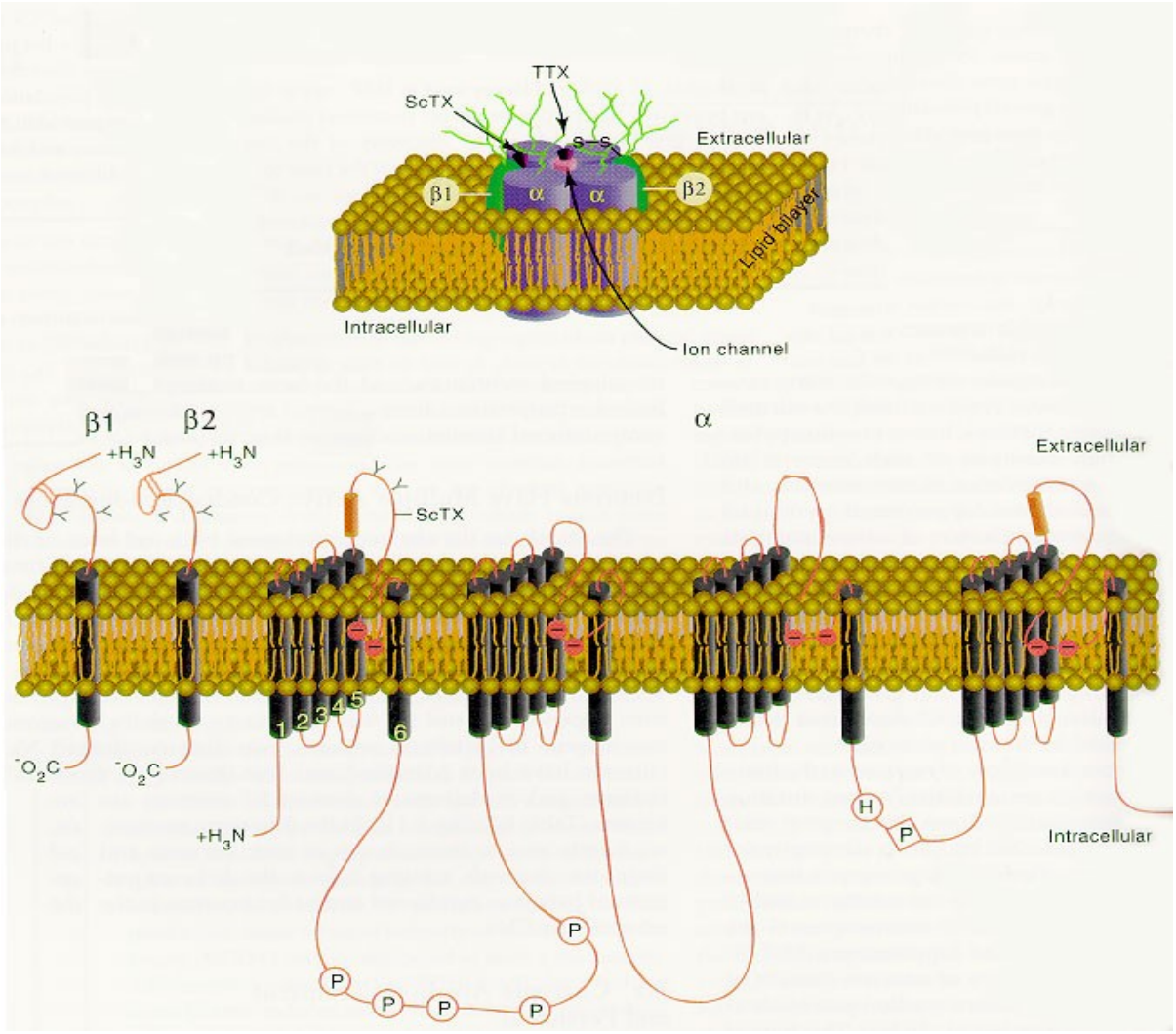


Lidocaine

CHEMICAL NAME:

N-diethyl aminoacetyl 2,6xylidine hydrochloride monohydrate. It contains a tertiary amine attached to an aromatic system by an intermediate chain. Tertiary amine is a base. Lignocaine is 65% protonated at PH 7.4.

SODIUM CHANNEL



Molecular weight of the base is 234 and that of hydrochloride salt is 270. Its Pka

is 7.9.

MECHANISM OF ACTION:

The action of the local anaesthetic is on the cell membrane of the axon, on which it produces electrical stabilization. The transient increase in the permeability of sodium ions necessary for propagation of the impulse is prevented, thus the resting membrane potential is maintained and depolarization in response to stimulation is inhibited.

They act by blocking the voltage gated sodium and potassium channels on the internal nerve cell membrane. The sodium channel protein is bell shaped with four transmembrane domains arrayed symmetrically around a central pore that splits into four passages that communicate between the intra and extracellular spaces.

The four homologous domains [I-IV] contain six transmembrane alpha-helices [S1-S6] and an inactivating particle connecting domains III and IV. The S5 and S6 segments and the short loops between them form the pore. The fourth helix [S4] has positively charged arginine or lysine residues at every third position and is regarded as the voltage sensitive region of the sodium channel. The local anaesthetic binding site is located in the pore lining transmembrane segment 6 of domain I,III and IV[8].

Three major conformational states of the sodium channel exist they are resting, open and inactivated.

In contrast to voltage gated sodium channels, potassium channels are a diverse family of membrane proteins with many subtypes, although their distribution in nerve cells is not clear, they fulfill a number of roles in peripheral nerves, such as establishing the resting membrane potential and accomplishing repolarisation[8].

Local anaesthetics bind to the sodium receptors on domain IV, loop S6 and cause blockade of these channels. The affinity is higher in the open or inactivated states than in the resting state. As a result the height of action potential is reduced, the firing threshold is elevated, the spread of impulse conduction is slowed and the refractory period lengthened. Finally nerve conduction is completely blocked.

Recent evidence has highlightened the many diverse actions of local anaesthetics. They also interact with G-protein coupled receptors, muscarinic receptors and endothelial nitric oxide.

Attachment of local anaesthetics to G-protein coupled receptors linked to lysophosphatalic acid attenuates neutrophil, macrophage and monocyte function.

Lignocaine has reduced the surface expression of adhesion molecules on

polymorphonucleocytes, reduced priming of PMN by cytokines, and reduced chemotaxis, lysozyme release and free radical production.[8, 17]

PHARMACODYNAMICS:

- *CARDIOVASCULAR SYSTEM:*

It stabilizes the electrical activity of any excitable tissue. It stabilizes the aberrant conduction and the automaticity in abnormal or damaged fiber and suppresses cardiac arrthymias. It is useful in the treatment of ventricular arrhythmias that occur following myocardial infarction or cardiac surgery.[20]

It causes vasoconstriction at low concentration and vasodilatation at higher concentration due to stimulation and inhibition of calcium release.

- *CENTRAL NERVOUS SYSTEM:*

It produces sedation, light headedness while sometimes anxiety and restlessness occur. With more marked toxicity circum oral numbness, muscle twitchings and visual disturbances can occur. Severe toxicity proceeds to convulsions and coma as a result of medullary depression.

PHARMACOKINECTICS[17, 20]:

Absorption is slow in regional anaesthesia where as when given intravenously, peak levels are reached immediately. It is metabolized in the liver-amide hydrolysis by microsomal enzymes, ring hydroxylation and dealkylation. Its metabolite monoethylglycine xylidide is moderately toxic and effective antiarrhythmic. Its volume of distribution is 911 and the clearance is 95 l/min.

Dosage and Administration:

- Cardiac arrhythmias:1-2 mg/kg IV bolus followed by an infusion at the rate of 4mg/min and reduced to 2mg/min over a period of 2-4 hours.
- To attenuate the cardiovascular response to intubation 1.5mg/kg 3 minutes prior to laryngoscopy.
- For treatment of status epilepticus: Initial injection of 2mg/kg followed by IV infusion at the rate of 6 mg/kg/hr for a maximum of 5 hours.[8]

CONTRAINDICATIONS

- Known hypersensitivity to local anaesthetics of amide group such as prilocaine, mepivacaine or bupivacaine.
- Adams-stroke syndrome, or severe degree of sinoatrial, or atrioventricular block.

SAFE DOSE:[8]

Without adrenaline: 3mg/kg.

With adrenaline : 7mg/kg.

AVAILABLE PREPARATIONS:

1) 5%(heavy)for spinal Anaesthesia.

2) 1% and 2% vial for Nerve blocks and Epidural Anaesthesia(with and without adrenaline)

3) Lignocaine hydrochloride 2%(without preservative) for intravenous use.

4) 4% Topical spray.

5) 2.5% Lignocaine in combination with 2.5% Prilocaine as EMLA.

6) 2.5 – 5 % ointment.

7) 2% Jelly.

8) 2% Viscous.

9) 10% suppositories.

10) 10% Aerosol.

11)5% Topical patch.

12)15%spray.

13)2%Lignacaine with adrenaline.

PHARMACOLOGY OF MAGNESIUM SULPHATE

PHARMACOLOGY OF MAGNESIUM SULPHATE

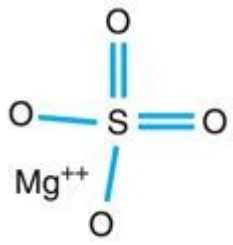
It is a bivalent ion like calcium with an atomic weight of 24.312.

Human body contains 1 mole (24g) of magnesium. It is the fourth common mineral salt in the body after phosphorus, calcium and potassium, second intracellular cation after potassium. In serum magnesium is divided into three fractions-

- Ionised,
- Protein bound and
- Contained in anion complexes.

These fractions account for 65%, 27%, and 8% in serum concentrations. respectively. [2, 12]

STRUCTURE OF MAGNISIUM SULPHATE[12]:



Magnesium sulphate

PROPERTIES OF MAGNESIUM SULPHATE:[2]

- ***CELLULAR PROPERTIES*:**

Magnesium intervenes in the activation of membrane calcium ATPase and Na - K ATPase involved in transmembrane ion exchange during depolarization and repolarization phases.It acts as a stabilizer of cell membrane and intracytoplasmic organelles.

- ***ION CHANNELS*:**

It acts as a regulator of different ion channels.It has a competitive antagonist action against calcium inflows there by limits the outflow of calcium from the sarcoplasmic reticulum.So it is a calcium channel blocker and calcium channel modulator. It is involved in hundreds of enzyme reactions in the body.

- ***CARDIOVASCULAR SYSTEM:***

It acts on calcium channels in the myocardial muscle and also acts directly on the cardiac muscle by inhibiting the calcium uptake on the troponin C of the myocytes and thereby influencing myocardial contractility.

Its vasodilatory action is due to its activation of cyclic AMP. This causes reduction in systolic blood pressure.

Coronary vascular resistance is reduced and causes vasodilatation but pulmonary vascular resistance is unaltered.

- ***NEURO MUSCULAR TRANSMISSION:***

It has a preponderant presynaptic and postsynaptic effect.

Magnesium acts competitively in blocking the entry of calcium into the presynaptic endings. Presynaptic release of acetylcholine is reduced by magnesium, thereby decreasing the effect of acetylcholine on the postsynaptic receptors, which in turn increases the threshold of axonal excitation.

It also produces progressive inhibition of catecholamine release from the adrenal medulla, adrenergic nerve endings and adrenergic post ganglionic sympathetic fibers.

- Acts as a antagonist of NMDA receptors, and this explains its use in post-op analgesia.
- Magnesium sulphate increases production of prostaglandins causing vasodilatation of the small intracranial vessels which is responsible for its anticonvulsant action.

- ***RESPIRATORY SYSTEM:***

It has bronchodilatory action due to the inhibition of smooth muscle contraction, histamine release from the mast cells and acetylcholine release from the cholinergic nerve endings.

PHARMACOKINETICS: [2, 17]

Absorption:

Following intravenous administration, onset of action is immediate and following intramuscular administration it takes about 1 hour for the onset of action.

Duration:

The duration of action following intravenous administration is about 30 minutes and following intramuscular route of administration the duration of action is about 3 to 4 hours.

Distribution:

Magnesium can cross placenta and also secreted in milk.

Elimination:

Metabolized in the liver and excreated through the kidneys; there is interindividual variability but the rate of excretion is directly propotional to the serum conecentration and glomerular filtration .

CLINICAL USES:

- For Severe Pre-eclampsia and Eclampsia: A loading dose of 4-6gm Magnesium sulphate diluted in 100ml of Normal Saline given over 15min intravenously. Then 2 gm/hr in 100ml of intravenous infusion (maintain serum levels between 4 and 7mEq/L).

Intermittent injection:

4gm magnesium sulphate is given slowly by intravenous route followed by 10gm,5gm in each buttock as deep intramuscular injection. Then every 4hrs 5gm intramuscularly upto24hrs after delivery.

- Magnesium sulphate has a tocolytic effect at serum levels of 8-10mEq/L. Loading dose of 4-6gm over 20min intravenously, then after the contraction ceases maintenance is done using 2-4gm per hour intravenously for 12-24 hours.

- To reduce the stress response during intubation-Magnesium sulphate is used in the dosage of 30-50mg/kg. intravenously.

- In surgery for phaeochromocytoma it helps to maintain haemodynamic balance because it inhibits the catecholamine release from adrenal medulla and adrenergic nerve endings.

- Nephritic Seizures: In children with nephritic seizures, the 50% concentration should be diluted to a 20% solution for intramuscular. injection. The dose for children is 20 to 40 mg (0.1 to 0.2 mL of a 20% solution)/kg of body weight, administered intramuscularly as needed, to control seizures.

- It is used postoperatively in patients who have undergone coronary artery bypass grafting to reduce the incidence of ventricular arrhythmias.

- It is also used in the treatment of Torsades De Pointes, as intravenously or intra osseously in the dosage of 25 to 50 mg/ kg (upto 2 gm).

- Acute Myocardial Infarction: Magnesium sulphate is used in the dose of 2gm intravenously over 5-15 min followed by 18 gm over 24hrs as infusion.

- Total Parenteral Nutrition: In total parenteral nutrition, maintenance requirements for magnesium are not precisely known. The maintenance dose recommended for adults is 5 to 8 mEq magnesium/L of total parenteral nutrition solution; typical daily adult intake ranges from 10 to 24 mEq. For infants, the recommended intake ranges from 0.25 to 0.6 mEq/kg/day.

- In barium poisoning: 1-2gm is used to counteract the intense muscle stimulating

effects of barium.

- In refractory bronchial asthma it is used for its bronchodilatory action.

- Hypomagnesemia: in case of mild deficiency 1gm every 6 hours for 4 doses, in severe cases 1-5gms (2 – 10ml of 50% solution) in divided doses, repeated until the serum levels are normal.

- Recent studies show its use in Tetanus patients, at a serum concentration of 2-4mEq/L, it gives good control of spasms and muscle rigidity.

PRECAUTIONS:

Because magnesium is removed from the body solely by the kidneys, the drug should be used with caution in patients with renal impairment. Urine output should be maintained at a level of 100 ml every 4 hours.

Monitoring serum magnesium levels and the patient's clinical status is essential to avoid the consequences of over dosage in toxemia.

Clinical indications of a safe dosage regimen include the presence of the patellar reflex (knee jerk) and absence of respiratory depression (approximately 16 breaths or

more/minute). Serum magnesium levels usually sufficient to control convulsions range from 3 to 6 mg/100 ml (2.5 to 5.0 mEq/L). The strength of the deep tendon reflexes begins to diminish when magnesium levels exceed 4 mEq/L.

Reflexes may be absent at 10 mEq magnesium/L, where respiratory paralysis is a potential hazard. An injectable calcium salt should be immediately available to counteract the potential hazards of magnesium intoxication in eclampsia.

Parenteral drug products should be inspected visually for particulate matter and discoloration prior to administration, whenever solution and container permit. Do not administer unless solution is clear and container is undamaged. Discard unused portion.

PREPARATIONS AVAILABLE:[2, 8]

Parentral injection:Magnesium sulphate-10%, 12.5%, 50%

For Intravenous use only-4%, 8%.

Magnesium sulphate in dextrose: 1% in 5%dextrose.

2%in 5% dextrose.

When administered intravenously the onset of action is immediate and duration of action is 30 min. on administration by intramuscular route the onset of action takes 1hr and duration of action is 3-4 hrs.

Storage: 15-30degree centigrade. For intravenous use concentration of 20% or less should be used. Rate of injection should be 1.5ml/hr.

DRUG INTERACTIONS:

Central Nervous System Depressants: When barbiturates, opiates, general anesthetics, or other CNS depressants are administered concomitantly with magnesium sulfate, dosage of these agents must be carefully adjusted because of the additive central depressant effects.

Neuromuscular Blocking Agents: Excessive neuromuscular blockade has occurred in patients receiving parenteral magnesium sulfate and a neuromuscular blocking agent; these drugs should be administered concomitantly only with caution.

Cardiac Glycosides: Magnesium salts should be administered with extreme caution in digitalized patients, because serious changes in cardiac conduction, which can result in heart block, may occur if administration of calcium is required to treat magnesium toxicity.[8]

ADVERSE REACTIONS:

The adverse effects of parenterally administered magnesium usually are the result

of magnesium intoxication. These include flushing, sweating, hypotension, depressed reflexes, flaccid paralysis, hypothermia, circulatory collapse, cardiac and CNS depression proceeding to respiratory paralysis. hypocalcaemia, with signs of tetany secondary to magnesium sulfate therapy for eclampsia, has been reported.

SYMPTOMS AND TREATMENT OF OVERDOSE:

Magnesium intoxication is manifested by a sharp drop in blood pressure and respiratory paralysis. Disappearance of the patellar reflex is a useful clinical sign to detect the onset of magnesium intoxication.

In the event of over dosage, artificial ventilation must be provided until a calcium salt can be injected intravenously to antagonize the effects of magnesium.

In adults, intravenous administration of 5 to 10 mEq of 10% calcium gluconate will usually reverse respiratory depression or heart block due to magnesium intoxication.

In extreme cases, peritoneal dialysis or hemodialysis may be required.

Hypermagnesemia in the newborn may require resuscitation and assisted ventilation via endotracheal intubation or intermittent positive pressure ventilation, as well as intravenous calcium. [17]

MATERIALS & METHODS

MATERIALS AND METHODS

After approval of the study by our institutional Ethics Committee, a total of 40 patients of both sexes in the age group between 15-50years, belonging to ASA grade I undergoing elective surgery under general anaesthesia were included in this double blinded, randomized, clinical study.

Informed written consent was obtained from all the patients. Patients were assessed by detailed history and physical examination, supported by routine investigations (Haemoglobin percentage, Blood glucose, urea, creatinine, ECG, X - ray chest PA view.)

INCLUSION CRITERIA are patients belonging to ASA grade – I, MPG grade – I, and laryngoscopy time less than 15 seconds.

EXCLUSION CRITERIA for the study includes patients belonging to ASA grade more than one., MPG > 1, predicted difficult airway, systemic hypertension, coronary artery heart disease, diabetes mellitus, patients on antihypertensives and cardiac drugs, and valvular heart diseases.

Out of the 40 patients 20 were randomly included in the L group (LIGNOCAINE GROUP) and the other 20 were included in the M group (MAGNESIUM SULPHATE

GROUP).

All the patients were premedicated with injection Glycopyrolate 0.01mg/kg intramuscularly 45minutes prior to surgery.

Patients were shifted to the operation theatre and connected to the noninvasive multimonitor, and intravenous access was obtained using 18G cannula.

Baseline Heart rate, Blood pressure (Mean arterial pressure), SPO_2 were recorded.

The patients were preoxygenated with 100% oxygen for 3 minutes. All the patients received injection fentanyl 1mcg/kg intravenously.

Two minutes after the administration of fentanyl, patients in L group received injection Lignocaine 1.5mg/kg and the patients in the M group received injection Magnesium sulphate 30mg/kg intravenously.

One minute after that the patients were induced with injection Thiopentone5mg/kg and injection atracurium 0.5mg/kg intravenously. Then the patients were mask ventilated with 100% oxygen and after 3 minutes heart rate, blood pressure (mean arterial pressure) were noted and taken as the post induction value.

Laryngoscopy and Intubation was done using appropriate size Macintosh blade and appropriate size endotracheal tube.

Measurement of heart rate, blood pressure (Mean arterial pressure) and SPO_2 was done immediately after laryngoscopy and at one, three and five minutes after placement of the endotracheal tube. Surgical incision was allowed after the last measurement.

Syringes were prepared by a postgraduate who did not take part in the study and the injections were given by him. Intubation was performed by an experienced anaesthesiologist who was double blinded to the drugs given.

Neuromuscular blockade was monitored using the nerve stimulator with train of four for every 5 minutes after intubation upto 45minutes.

The incidence of complications like hypotension, arrhythmias, nausea, flushing, sweating were recorded until the patient was discharged from the post anaesthesia care unit.

RESULTS

RESULTS

The present study was undertaken in 40 ASA grade I patients of both gender between the age group of 15-50 years scheduled for elective surgeries under general anaesthesia. The patients were categorized into 2 groups [Lignocaine group and Magnesium group].

Statistical Analysis

We used students independent "t" test to compare various factors between the two groups. Results were expressed as mean and standard deviation (Mean ± SD) Chi-square test was done to compare proportions, and values less than 0.05 was considered as statistically significant.

(Table: 1) (Figure: 1)

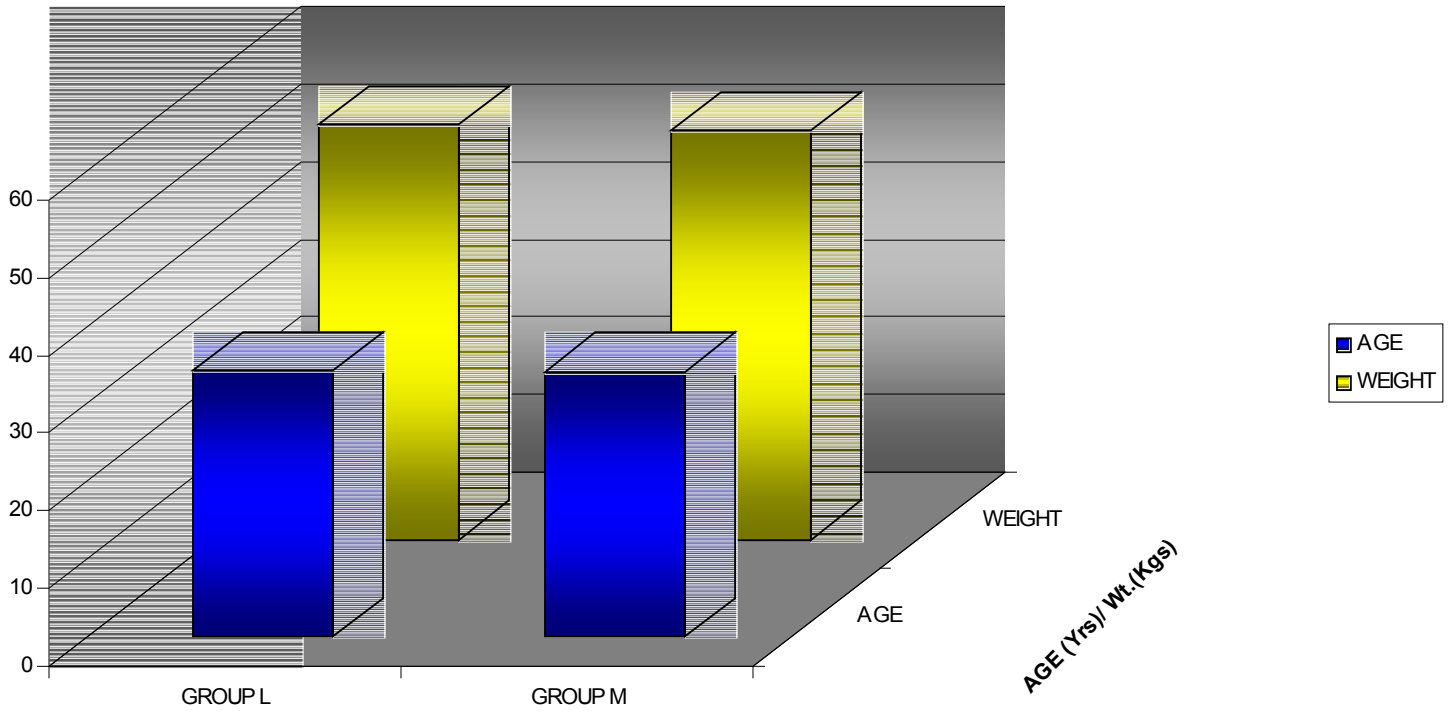
Table: 1 **DEMOGRAPHIC CHARACTERISTICS BETWEEN GROUPS**

PARAMETERS	**GROUP –L (n=20)**	**GROUP – M (n=20)**	**P value**
Mean Age in Yrs (±S.D.)	34.3 ± 8.86	34.2 ± 8.22	0.97
Mean weight in Kgs (±S.D.)	53.5 ± 3.38	52.8 ± 4.40	0.54

(S.D. – Standard deviation) **(P > 0.05)**

Figure: 1

DEMOGRAPHIC DATA



The groups were matched for demographic data, and there was no statistically significant difference found between the groups in age, sex, weight and surgical position.

With patient on table before giving Lignocaine or Magnesium sulphate, baseline heart rate and mean arterial pressure were recorded for the two groups (Table: 2) and (Table: 3) respectively. There was no significant difference in heart rate and MAP between the 2 groups.

Table: 2 **BASELINE HEART RATE.**

BASELINE HR	*GROUP – L*	**GROUP – M**
MEAN ± S.D.	86.8 ± 5.063	87.25 ± 4.529

(HR – Heart rate) (P > 0.05) Figure :2

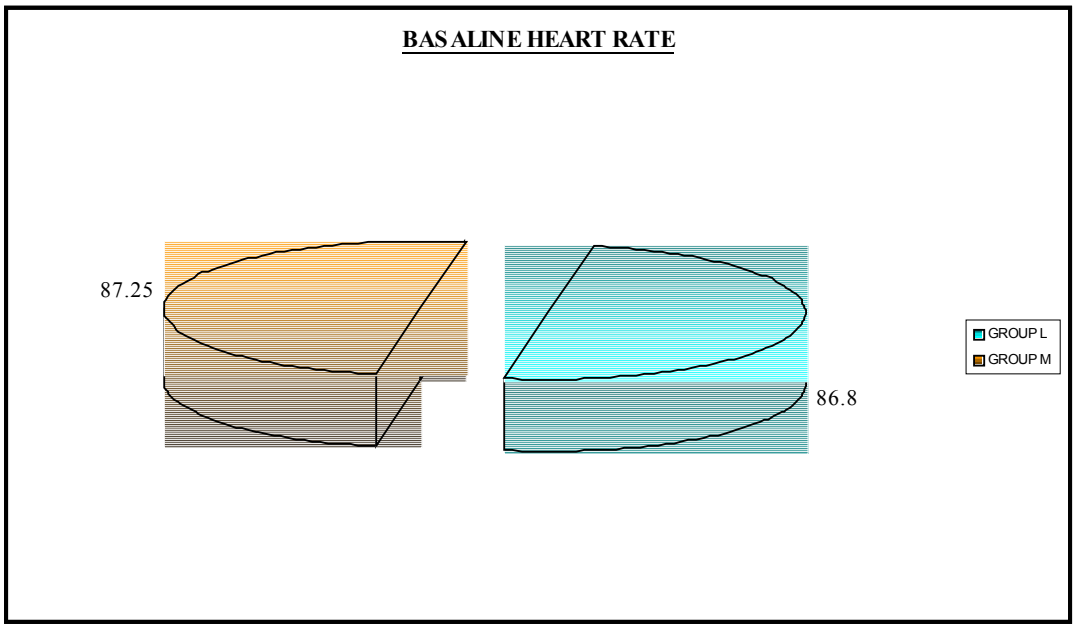


Table : 3 **BASE LINE MEAN ARTERIAL PRESSURE.**

BASELINE MAP	GROUP – L	GROUP – M
MEAN ± S.D.	93.6 ± 10.1	95.9 ± 9.49

(MAP – Mean Arterial Pressure) (P > 0.05) Fig.3

After the drug was administered intravenously any deviation from the baseline values were recorded following induction, laryngoscopy, and intubation. (1 minute, 3 minutes, and 5minutes) (Table: 4, Table: 5)

Figure: 3

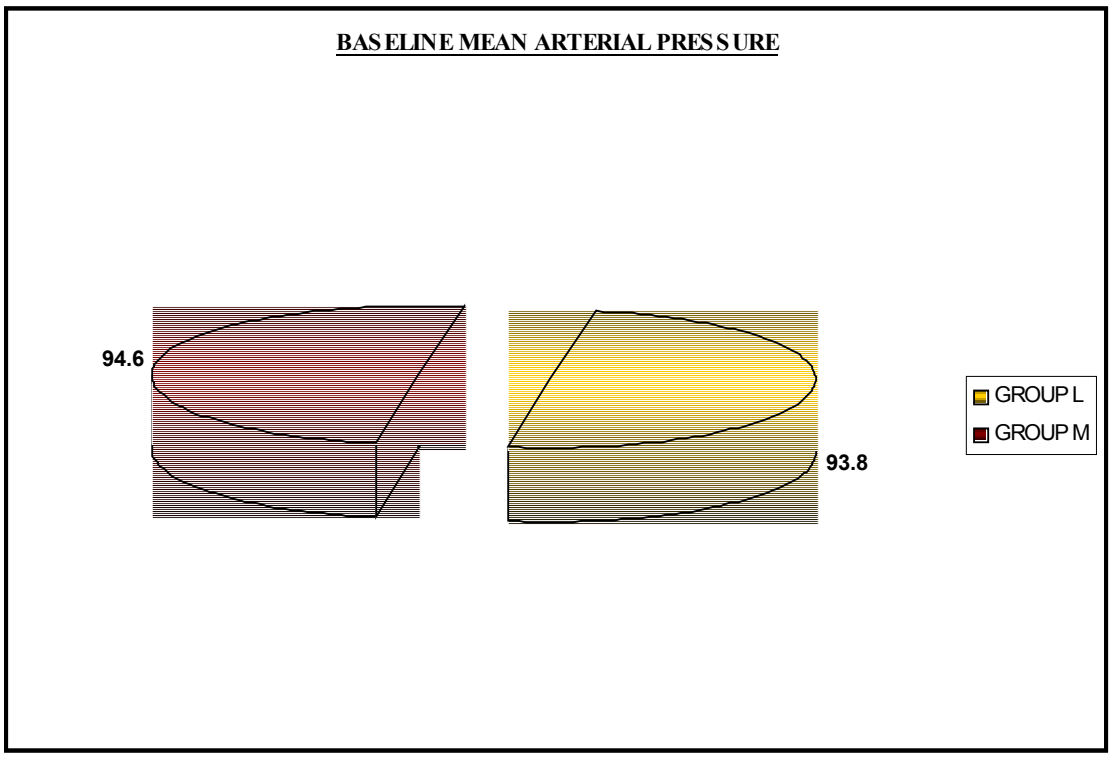


Table: 4 **DEVIATION OF HEART RATE FROM BASELINE**.

HEART RATE	GROUP – L MEAN ± S.D	GROUP – M MEAN ± S.D.	P VALUES
POST INDUCTION	80.85 ± 5.31	75.8 ± 3.95	0.002
POST LARYNGSCOPY	84.4 ± 4.75	78 ± 4.18	0.0001
POST INTUBATION (1 min.)	86.2 ± 4.77	79.3 ± 4.02	0.0001
POST INTUBATION (3 min.)	84.4 ± 4.82	77.4 ± 4.19	0.0001
POST INTUBATION (5 min.)	83 ± 4.36	75.8 ± 4.14	0.0001

(P < 0.005)

There was fall in heart rate (P < 0.05) noted following induction, laryngoscopy, and intubation, in Magnesium group and Lignocaine group, but the fall in heart rate was more significant in the Magnesium group compared with the Lignocaine gr

DEPICTION OF HEART RATE

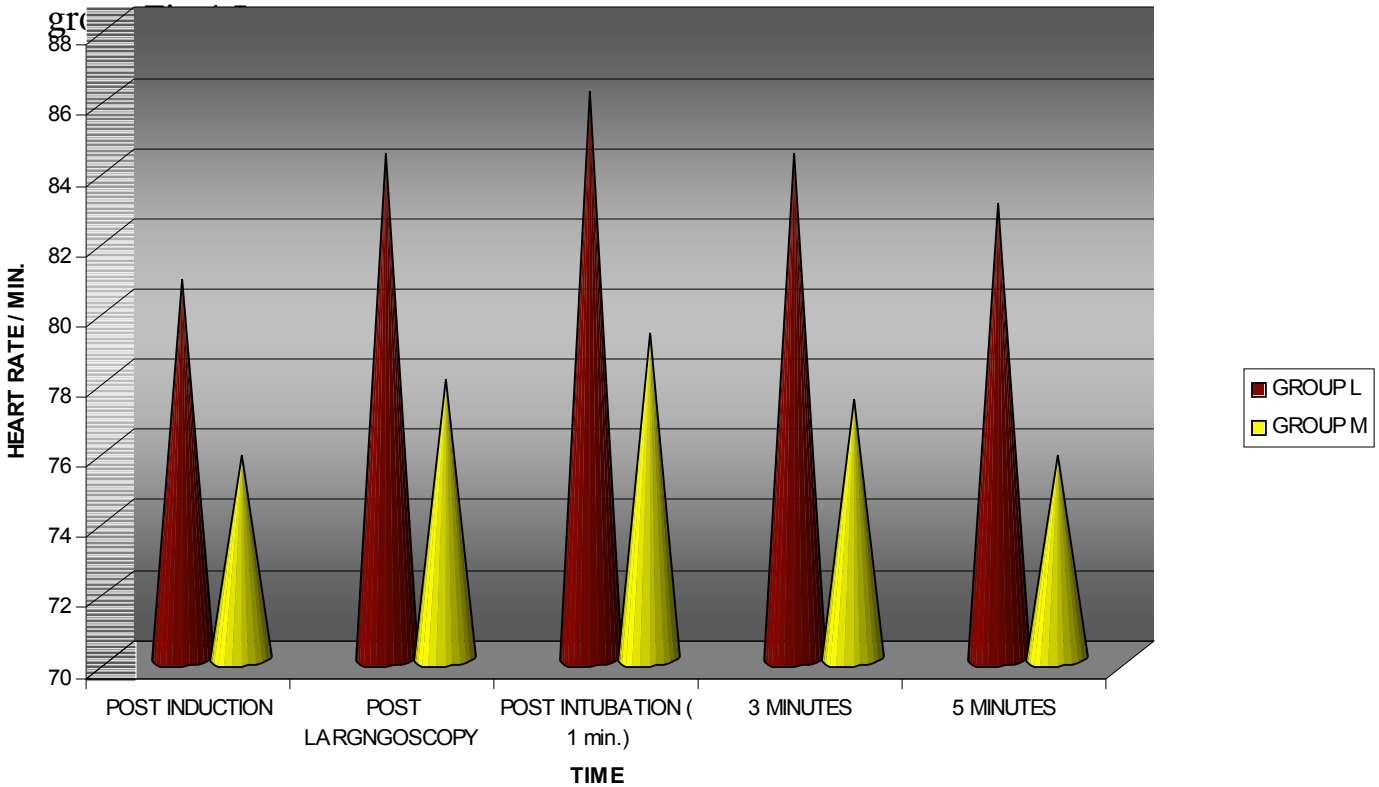


Figure: 5

COMPARISON OF HEART RATE

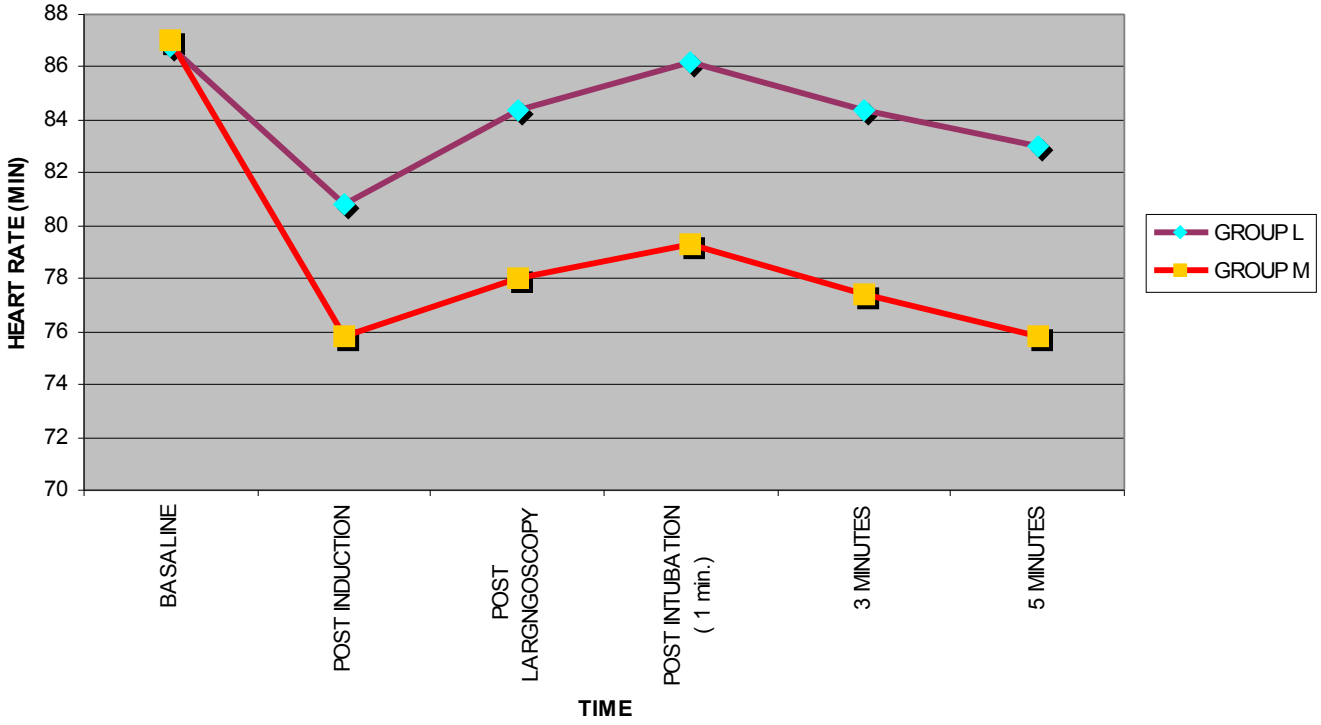


Table : 5 **DEVIATION OF MEAN ARTERIAL PRESSURE FROM BASELINE.**

MAP	GROUP – L MEAN ± S.D	GROUP – M MEAN ± S.D.	P VALUES
POST INDUCTION	87.7 ± 9.35	81.8 ± 17	0.021
POST LARYNGSCOPY	91.3 ± 9.23	85.6 ± 7.17	0.035
POST INTUBATION (1 min)	92.7 ± 10	86.7 ± 6.78	0.032
POST INTUBATION (3 min)	91.2 ± 9.84	85 ± 6.92	0.028
POST INTUBATION (5 min.)	90.2 ± 9.96	83.5 ± 6.65	0.017

(MAP – Mean arterial pressure) (P < 0.05)

Like heart rate there was a significant fall in mean arterial pressure in both the groups, but there was statistically significant fall (P < 0.05) in the Magnesium group when compared with the L group in MAP. Fig 6.7

DEVIATION OF MEAN ARTERIAL PRESSURE

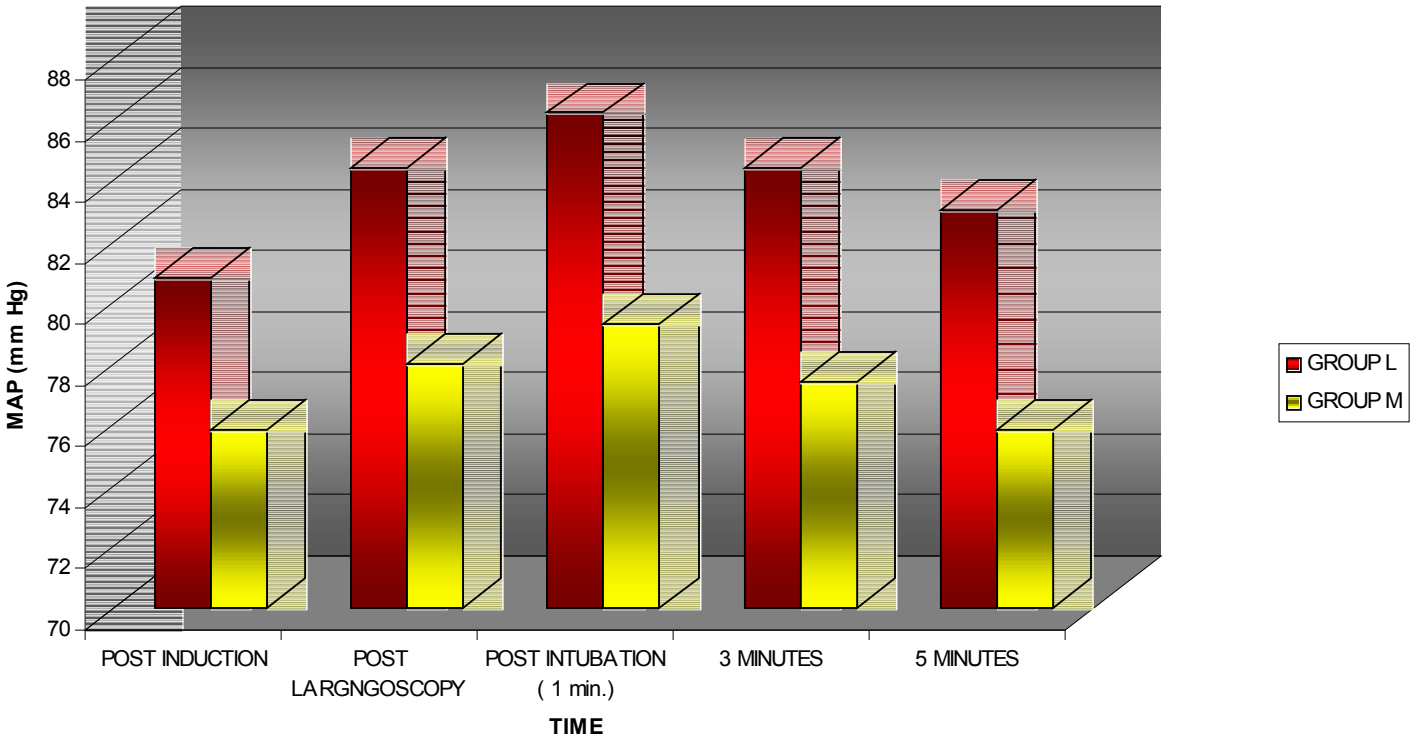


Figure: 7

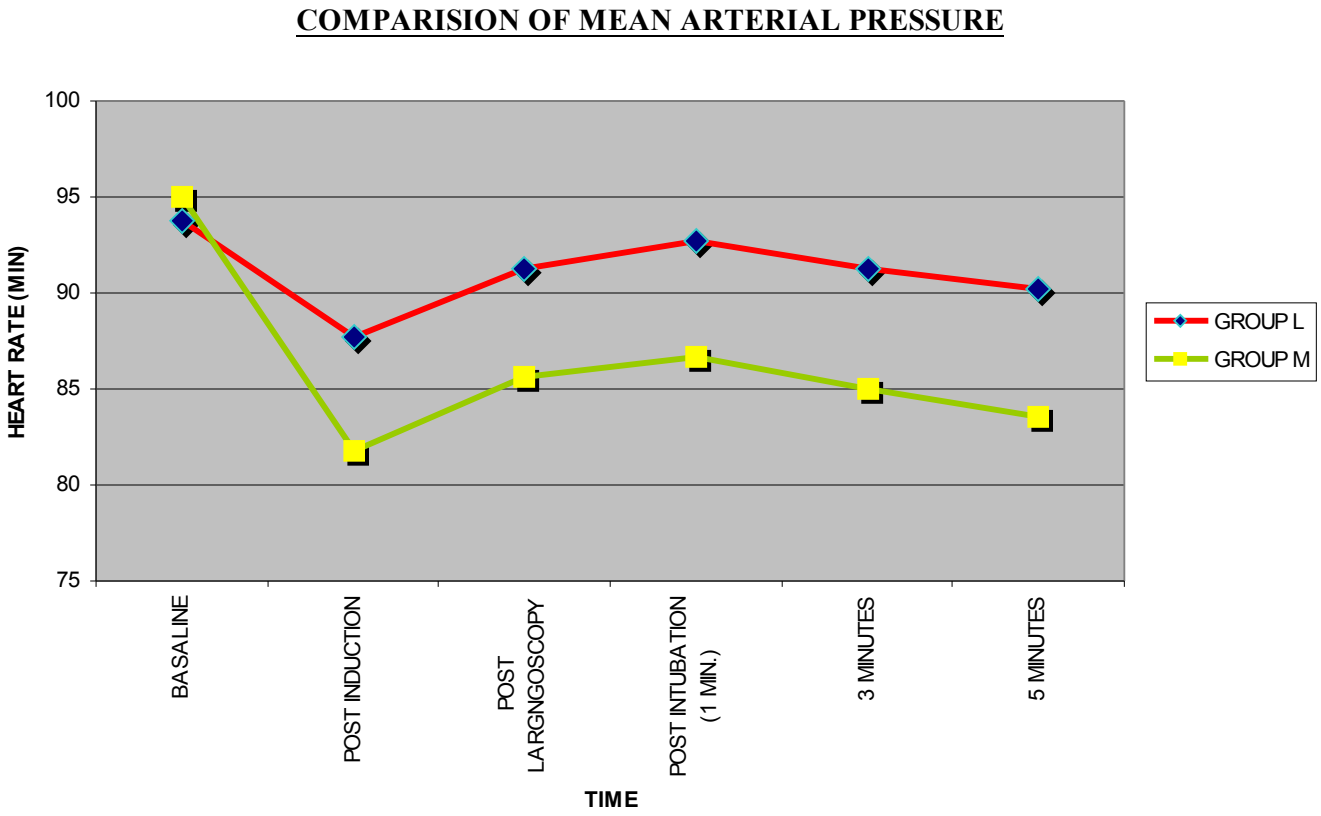


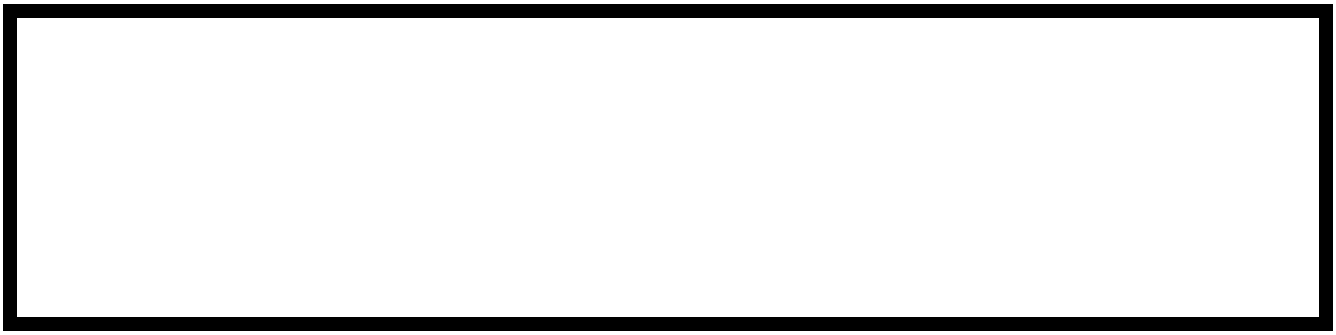
Table : 6 **AVERAGE VALUES OF TRAIN OF FOUR.**

TOF IN MINUTES	GROUP – L	GROUP – M	P VALUE
MEAN ± S.D.	25.5 ± 4.84	26.5 ± 4.89	0.52

(P > 0.05)

The train of four values was recorded every 5 minutes upto 45 minutes following drug administration and the duration needed for the return of third twitch using neuromuscular monitoring was recorded. This showed no statistically significant difference between the 2 groups. (P >0.05). (Table: 6)

The incidence of complications for magnesium like hypotension, sweating, arrhythmia, nausea, flushing, and hot sense were watched for until the patients were discharged from post anaesthesia care unit. There were no such complications observed in this study.



REVIEW OF LITERATURE

REVIEW OF LITERATURE

The haemodynamic consequences of endotracheal intubation have been the subject of study by various authors over many years

Tachycardia and hypertension commonly occur during anaesthesia due to many factors like mechanical stimulation, abnormal level of electrolytes and complex interaction of hypoxia, hypercapnia and circulating levels of catecholamines.[17]

Gray & Nunn[22]n their study proved "Laryngoscope and intubation are the commonest cause of transient hypertension and arrhythmias during anaesthesia. Fortunately the arrhythmias normally disappear once the tube is in place."

King et.al[16]*. in 1951.*showed that there was a marked rise in blood pressure and heart rate during laryngoscopy which was due to the mechanical stimulation of sensitive receptors in the area of epiglottis.

Baumgartner & Mycoft concluded in their study that "Succinyl choline produces transient rise in blood pressure accompanied by bradycardia"

Collins et.al. [17] proved that " Deep pressure on the base of the tongue and on the neck muscle is responsible for the rise in blood pressure and heart rate .

Chung et.al. [20]in their study on haemodynamic responses to laryngoscopy and intubation showed maximum value of systolic , diastolic , mean arterial pressure and heart rate occur within 30 to 60 seconds of laryngoscopy and intubation, and in patients with hypertension, laryngoscopy and intubation led to left ventricular failure due to exaggerated pressure response. Similarly in patients with IHD transient myocardial ischaemia has been observed.

Aries & simonies [18]have correlated the levels of adrenaline and nor adrenaline with sympathetic innervation."

Roberts, C. Green, et. al [22]showed an exaggerated form of rise in heart rate and mean arterial pressure in hypertensive patients during laryngoscopy and intubation.

A.J. Shribiran, G. Smith et. al.[19] in a study of haemodynamic stress response to laryngoscopy alone and laryngoscopy followed by intubation, was done in 24 patients and the heart rate, MAP, and the plasma catecholamines before laryngoscopy and at 1st, 3rd, and 5th min after laryngoscopy was assessed. There was a significant increase in the heart rate, mean arterial pressure and plasma catecholamine concentration following laryngoscopy with or without intubation.[19]

R.W.Allen,M.B,CH.B,F.F.A.R.C.S.I,and M.F.Mjames,P.C.Uys[6] in their study on attenuation of the pressor response to tracheal intubation in hypertensive proteinuric pregnant patients they compared lignocaine and magnesium sulphate 40mg/kg in 69 patient and found that the attenuation of heart rate and MAP was more significant in the Magnesium group.[6]

Similarly *D.H.Van ziji,P.C.Gordon and M.F.James*[20]ompared the effects of Remefentanil or Magnesium sulphate with placebo in attenuating the haemodynamic response after electroconvulsive therapy and found significant response with magnesium sulphate.[3]

Michael F. M. James, FFARCS, R. Eryk Beer, FFA(SA), and Jan D. Esser[3], MMED **in their study on** "Intravenous Magnesium Sulfate Inhibits Catecholamine Release Associated with Tracheal Intubation" showed pretreatment with Magnesium sulphate 60mg/kg in 15 patients compared with normal saline found that Magnesium group patients had decrease in heart rate and mean arterial pressure and the plasma levels of epinephrine was unchanged after intubation.[3]

Puri GD et. al [4]showed the effect of magnesium sulphate on haemodynamics and its efficacy in attenuating the response to endo tracheal intubation in patients with coronary artery disease, concluded magnesium administration at the time of the induction of anesthesia improves hemodynamics in patients with CAD undergoing CABG and is associated with lesser hemodynamic and ST segment changes compared with lidocaine at the time of endotracheal intubation in these patients. When administering magnesium sulfate, however, one should be aware of the adverse hemodynamic interactions inpatients with CAD receiving calcium channel blockers, B-adrenergic blockers and ACE inhibitors for antihypertensive therapy.

N. M. Elsharnouby and M. M. Elsharnouby[13] in their paper on Magnesium sulphate as a technique of hypotensive anaesthesia studied sixty patients (25 female) undergoing functional endoscopic sinus surgery in two parallel groups. The magnesium group received Magnesium sulphate 40 mg kg^{-1} i.v. as a bolus before induction of

anaesthesia and 15 mg kg^{-1} h^{-1} by continuous i.v. infusion during the operation. The same volume of isotonic solution was administered to the control group. Intraoperative bleeding was evaluated using a quality scale, showed Magnesium sulphate led to a reduction in arterial pressure, heart rate, blood loss and duration of surgery. Furthermore, magnesium infusion alters anaesthetic dose requirements and emergence time

G M Sanders, FRCA, K M Sim, M Med (Anaes)[15] in their study

Is it Feasible to Use Magnesium Sulphate as a Hypotensive Agent in

Oral and Maxillofacial Surgery?

They have demonstrated that the use of intravenous Magnesium sulphate infusion for intraoperative deliberate hypotension is feasible in ASA 1 patients undergoing standard nitrous oxide, oxygen, isoflurane, opioid and muscle relaxant anaesthesia. Blood loss appears to be less than with other hypotensive anaesthetic techniques. Intraoperative control of blood pressure was satisfactory without undesirable cardiovascular side effects and postoperative muscle weakness was not a problem clinically.

In a Dose – Response Study of Magnesium Sulfate in Suppressing

Cardiovascular Responses to Laryngoscopy and Endotracheal Intubation.

By *K. Montazeri MD, M. Fallah MD.*[1] on 120 ASA – 1 patients classified into 6 groups of 20 each Magnesium sulphate was given in the doses of 10, 20, 30, 40, 50mg/kg each and compared with lignocaine 1.5mg / kg. The induction of anesthesia was same in all groups and the pulse rate and arterial blood pressure were measured and recorded just before intubation and also at 1, 3, and 5 minutes after intubation before surgical incision.

In their conclusion they proved that administration of different doses of Magnesium sulphate intravenously at the time of the induction of anesthesia improves hemodynamic responses to endotracheal intubation. They found that the dose of 30mg/Kg of Magnesium sulphate was most effective with least adverse effects.

On intravenous Magnesium sulfate as a preanesthetic medication:

Tetsuro Kagawa et. al.[11] in their double-blind study on its effects on hemodynamic stabilization at the time of tracheal intubation showed the effects of Magnesium sulfate ($MgSO_4$) as a preanesthetic medication with regard to whether it can sedate or relieve a patient who is scheduled to undergo surgery, and whether it can control the hemodynamic response to tracheal intubation. Twenty adult patients in ASA status I–II undergoing elective surgery were studied. Magnesium sulphate was given in dose of 50 mg/kg intravenously by drip infusion from 30 min before the induction of anesthesia and

saline was used as a control. The changes in mean arterial pressure (MAP) and rate pressure product (RPP) after the intubation were significantly suppressed in magnesium-treated patients, but a sedative effect was not observed in their study. Therefore, they concluded Magnesium sulphate was useful as a preanesthetic medication in suppressing the hemodynamic response associated with tracheal intubation.

Naghibi KH, Akhtari M et. al.[7] in their study of attenuation of pressor responses to trachaeal intubation by Magnesium sulphate, showed a reduction in mean arterial pressure and tachycardic response in patients who were pre treated with magnesium sulphate intravenously, with very low sedative effect.

DISCUSSION

DISCUSSION

Calcium exerts a major role in stimulus-response relationship, including the release of catecholamines from the adrenal gland and adrenergic nerve terminals in response to sympathetic stimulation. Because Magnesium competes with calcium for membrane channels, it has been described as the physiological calcium antagonist and can modify many calcium mediated responses.[10]

The ability of magnesium ions to inhibit the release of catecholamines from both the adrenal glands and peripheral adrenergic nerves terminals has been known for many years. It also produces vasodilatation, directly.

G.D. Puri et.al. [4]n their study on the effect of Magnesium sulphate on haemodynamics and its efficiency in attenuating the response to endotracheal intubation in patients with coronary artery disease, studied 36 patients, of which one group received 50mg/kg of Magnesium sulphate intravenously and another group received 1mg/kg Lignocaine intravenously.

Their results showed a decrease in mean arterial pressure from basal values of 91±

14.5 to 86 .6± 14.5 mm of Hg after intubation (P<0.05). The heart rate showed a mild increase from 65.2± 12.7 to 69.7± 13.7 beats/ minute (P < 0.001), and the cardiac index is also increased (P< 0.01) in the Magnesium sulphate group when compared with control group.

Three patients in the magnesium group and two patients in the control group had severe hypotension that needed pharmacological treatment. This fall was noted in the patients who were taking ACE inhibitors or beta-blockers for coronary artery disease and this potentiated the myocardial depressing effect of Magnesium sulphate. In this study all the patients were ventilated post operatively, so the enhancing effect of neuromuscular blockade was not studied.

On comparison with our study, in which we used Magnesium sulphate in the dose of 30mg/kg intravenously to asses the cardiovascular response to laryngoscopy and intubation showed a decrease in heart rate (P < 0.005) and the mean arterial pressure also showed a decrease (P < 0.05) on comparison with the Lignocaine group.

The increase in the heart rate noted in this study by G.D.Puri et.al.[4] may be due the ability of Magnesium sulphate in higher doses to inhibit the release of acetylcholine from the vagal nerve predominantly. This theory was supported by Michael F.M. James, FFARCS, R. Eryk Beer, FFA(SA), and Jan D. Esser, MMED[24] in their study, “

Intravenous Magnesium sulphate inhibits catecholamine release associated with tracheal intubation" in which they used Magnesium sulphate in the dose of 60mg/kg intravenously and their results showed an initial increase in heart rate by 13± 3.9 beats/ minute.

This effect was not observed in our study as we used a dose of 30mg/kg, which was a lesser dose on comparison with their study.

The mean arterial pressure in both studies showed a declining trend, but three patients in the Magnesium sulphate group in their study showed hypotension which required treatment, which was not observed in our study since we included ASA grade I patients, who were not on any drugs acting on cardiovascular system preoperatively. However we should be aware of the adverse haemodynamic interactions of Magnesium sulphate in patients receiving cardiovascular drugs like ACE inhibitors, betablockers and calcium channel blockers.

In a dose response study of Magnesium sulphate in suppressing the cardiovascular responses to laryngoscopy and intubation done by K.Montazeri M.D., M.Fallah M.D.[1] studied 6 groups of patients of 20 each with varying doses of Magnesium sulphate (10,20,30,40,and 50mg/kg) and compared with the control using Lignocaine in the dose of 1.5mg/kg. and observed that there was significant reduction in heart rate on

comparison between Magnesium sulphate and Lignocaine groups ($P < 0.05$). But within the magnesium groups the difference in heart rate was not significant ($P > 0.05$).

Like heart rate there was significant fall in the mean arterial pressure when compared with the lignocaine group ($P < 0.05$), but the fall is not significant within the magnesium groups ($P > 0.05$) .

The train of four at 45 minutes after induction of anaesthesia in all groups had no statistically significant differences.

Adverse effects of Magnesium sulphate like hypotension, arrhythmia, nausea, sweating and flushing, were observed in certain patients in higher doses.

Similarly in our study using 30mg/kg of Magnesium sulphate there was a reduction in the heart rate ($P <0.05$) and the mean arterial pressure ($P<0.05$), which was statistically significant when compared with the lignocaine group lending support to our study. We did not observe any side effects in any of the patients. Similarly neuromuscular blockade was not significantly prolonged in our study as seen by the train of four values at 45 minutes.

In a study by G.M. Saunders FRCA, K.M. Sim M.Md (Anaes),[15] " Is it feasible

to use Magnesium sulphate as hypotensive agent in oral and maxillo facial surgery?" studied 16 patients using Magnesium sulphate infusion at 40g/hr. until the mean arterial pressure reached to 55± 5 mm Hg and followed by maintenance of 5g/hr until 30 min. prior to the end of surgery, their results showed decrease in mean arterial pressure and heart rate from a baseline during the surgery and which returned to baseline 26 minutes after the surgery. Train of four monitoring during surgery showed no response in all the patients on Magnesium sulphate infusion. On terminating the Magnesium sulphate infusion the first tetanic contraction appeared with no fade, followed by train of four pattern similar to that seen with depolarizing neuromuscular blockers. There were no complications like reflex tachycardia or arrhythmias observed.

This study corroborates with our study in the reduction of mean arterial pressure and heart rate significantly in intravenous use of Magnesium sulphate, but we altered the dose to 30mg/kg bolus, as they have used higher doses and observed few side effects like prolonged sedation postoperatively.

In another study by R.W. Allen,M.B., CH.B., F.F.A.R.C.S.I, M.F.M. James, CH.B., P.C. Uys, CH.b. FFA (SA)[6] in " Attenuation of the pressor response to tracheal intubation in hypertensive proteinuric pregnant patients by Lignocaine, Alfentanil, and Magnesium sulphate". they used Magnesium sulphate 40mg/kg intravenously and observed an increase in the systolic, diastolic blood pressure and mean arterial pressure

following intubation, in the lignocaine group when compared to the Magnesium group. Similarly the heart rate was decreased in the Magnesium sulphate group significantly when compared to the other two groups. Alfentanil caused least change in heart rate but caused significant fetal depression. Although Magnesium sulphate and Alfentanil provide adequate control of cardiovascular response in hypertensive patients, Alfentanil is less reliable in controlling severe hypertension.

This study corroborates with the evidences in our study that Magnesium sulphate intravenously given prior to induction can have adequate control of cardiovascular response to laryngoscopy and intubation.

Our present study lends support to previous studies that the use of Magnesium sulphate 30mg/kg intravenously one minute prior to induction reduces the heart rate and mean arterial pressure in response to laryngoscopy and intubation in a favourable manner without any side effects.

SUMMARY

SUMMARY

This study was conducted in 40 ASA grade I patients who were admitted at Thanjavur Medical College Hospital to undergo various surgical procedures under general anaesthesia After getting Ethical committee approval and informed written consent from the patients, the total of 40 patients were allocated into two groups of 20 each.

They were connected to the non-invasive monitors and the basal heart rate and mean arterial pressure were recorded.

Magnesium sulphate was given in the dose of 30mg/kg one minute prior to induction for group M and Lignocaine 1.5mg/kg was given to group L.

The induction of anaesthesia was same in both groups and heart rate and mean arterial pressure were recorded following induction, laryngoscopy, and intubation at one, three, and five minutes (before the surgical incision). Train of four was recorded for 45 min. from the time of induction for every 5 minutes until the third twitch appears in the neuromuscular monitoring. Patients were observed for side effects of Magnesium sulphate like hypotension, arrhythmias, nausea, flushing and sweating until they were

discharged from post anaesthesia care unit.

The results were analysed using student t –test, and a P value of less the 0.05 was taken significant.

With patients matched for demographic data the results showed there was no significant difference in base line values between two groups. There was a reduction in the heart rate and mean arterial pressure in both groups but when both the groups were compared there was statistically significant reduction of heart rate and mean arterial pressure in Magnesium sulphate group. ($P < 0.05$). There was no significant prolongation of Train of four values between the two groups, and there was no side effects observed in any of the patients in this study.

CONCLUSION

CONCLUSION

We conclude that, Magnesium sulphate in the dose of 30mg/kg given intravenously one minute prior to induction,

- Attenuates the cardiovascular responses to laryngoscopy and intubation in a better manner than lignocaine.

- Does not cause any adverse effects in any of the patients.

- Does not cause prolongation of the neuromuscular blockade.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. **K. Montazeri M.D., M. Fallah M.D.,** A Dose – Response study of Magnesium sulphate in suppressing the cardiovascular responses to laryngoscopy and endotracheal intubation. Journal of Research in Medical Sciences 2005; 10(2): 82 – 86.

2. **Fawcett WJ, Haxby EJ, Male DA**. Magnesium: physiology and pharmacology. J Anaesth1999 Aug;83(2):302-20.

3. **James MF, Beer RE, Esser JD**. Intravenous magnesium sulfate inhibits catecholamine release associated with tracheal intubation. Anesth Analg 1989 Jun;68(6):772-6.

4. **Puri GD, Marudhachalam KS, Chari P, Suri RK.** The effect of magnesium sulphate on haemodynamics and its efficacy in attenuating the response to endotracheal intubation in patients with coronary artery disease. Anesth Analg1998 Oct;87(4):808-11

5. **Naghibi KH, Akhtari M**. Attenuation of the pressor responses to tracheal intubation by MgSO4. Res med sci J 2000;Supp 1:42-44.

6. **Allen RW, James MF, Uys PC.** Attenuation of the pressor response to tracheal intubation in hypertensive proteinuric pregnant patients by lignocaine, alfentanil and magnesium sulphate. Br J Anaesth 1991 Feb;66(2):216-23.

7. **Naghibi KH, Akhtari M.** Attenuation of the pressor responses to tracheal intubation by MgSO4. Res med sci J 2000;Supp 1:42-44.

8. AHFS. Drug information. American society of health system pharmacists; 2000: 2169-4.

9. **Kaye AD, Grogono AW.** Fluid and Electrolyte physiology. In: Miller RD. Anesthesia. 5th edition. Philadelpphia: Churchill Livingstone; 2000: p1593- 1594

10. **Rasmussen H.** The calcium messenger system (2). N Engl J Med1986 May 1; 314(18):1164-70.

11. **Tetsuro Kagawa 1, Ryokichi Goto 1, Katsuhiro Iijima], Yuji Kamikawa, Kenchi Sakai , and Hidefumi Obara .** Intravenous magnesium sulfate as a preanesthetic medication: A double-blind study on its effects on haemodynamic stabilization at the time of tracheal intubation. J A.nesth (1994) 8:17-20.

12. The dynamic structure of the sulfate ion in aqueous solution Studied by Raman Bandshape analysis **D. Watanabe and H. Hamaguchi.** Department of Chemistry, School of Science, The University of Tokyo; Lett. 339, 351 (2001).

13. **N. M. Elsharnouby' and M. M. Elsharnouby;** Magnesium sulphate as a technique of hypotensive anaesthesia. British Journal of Anaesthesia 2006 96(6):727-731.

14. ANATOMY FOR ANAESTHETISTS, **Harold Ellis**, CBE, MA, DM, FRCS, FACS (Hon). ;p 17 – 37.

15. Is it Feasible to Use Magnesium Sulphate as a Hypotensive Agent in Oral and Maxillofacial Surgery?, **G M Sanders,FRCA, K M Sim, M** Med (Anaes); Ann Acad Med Singapore 1998; 27:780-5.

16. **King B.D., L.C. Harris, F.E. Greitenstein, J.D. Elder, and R.D. Dripps** – Reflex circulatory response to direct laryngoscopy and tracheal intubation performed during general anaesthesia – Anaesthesiology 1951, 12; 556-62.

17. **Miller's Anaesthesia** – 6th edition – Ronald D. Miller, p 1634.

18. **Aries and Simonis** Biochem. pharmacol.: 32, 1539, 1983.

19. **A.J. Shribiran, G. Smith et. al.**, - Cardiovascular and catecholamine responses to laryngoscopy with and without intubation,- British Journal of Anaesthesia, 1987, 59; 245 – 9.

20. **Wyle and Churchill Davidson** – A Practice of Anaesthesia; 7th edition.

21. **Robert K. Stoelting** – Circulatory changes during direct laryngoscopy and intubation – Anaesthesiology 1997; 47; 381 – 83.

22. **Prys Roberts, C Green** – Studies of anaesthesia in relation to the hypertension – Haemodynamic consequences of laryngoscopy and intubation – BJA; 1971, 43; 531 – 46.

23. **N. G. Lavies, B. H. Mechel Hohn, A.E. May, K.J. Achola and D. Fell:** Hypertensive and catecholamine response to tracheal intubation in patients with pregnancy induced hypertension, BJA. Vol.63. No. 4, 429 – 434; October 1989.

24. **Michael F. M. James, FFARCS, R. Eryk Beer, FFA(SA), and Jan D. Esser, MMED, "**Intravenous Magnesium Sulfate Inhibits Catecholamine Release Associated with Tracheal Intubation", Anesth Analg 1989; 68:772-776.

PROFORMA

PROFORMA

DEPARTMENT OF ANAESTHESIOLOGY
THANJAVUR MEDICAL COLLEGE

Name & Address: Age/ Sex: I.P. Number:

Assessment no. : Unit:

Diagnosis: Surgical procedure:

Anaesthesiologist: Surgeon:

PREANAESTHETIC ASSESMENT:

History:

CLINICAL EXAMINATION: P.R.: B.P: Ht.: Wt.:

CVS: RS: Airway:

ASA :

INVESTIGATIONS : Blood group: Hb%:

Urea Sugar ECG:

Blood Urine

Sugar Albumin CXR:

PREMEDICATION:

Sl. No:	***DRUG***	***DOSE***	***ROUTE***	***TIME***
1.				

PARAMETERS:

PARAMETERS	**HEART RATE (min)**	**SYSTOLIC BP (mm Hg)**	**DIASTOLIC BP (mm Hg)**	**MAP (mm Hg)**
BASELINE				
POST INDUCTION				
POST LARNYGSCOPY				
POST INTUBATION(1min)				
POST INTUBATION(3min)				
POST INTUBATION(5min)				

TOF:

INTRAOPERATIVE COMPLICATIONS:

PACU OBSERVATIONS:

MASTER CHART (LIGNOCAINE GROUP)

Sl. No.	Name	Age/ Sex	Wt. (kgs)	Basal		Post induction		Post laryngoscopy		Post intubation (1 min)	
				PR	MAP	PR	MAP	PR	MAP	PR	MAP
1	Maniammal	18/F	52	85	95	80	88	84	90	84	94
2	Chellammal	22/F	54	90	112	84	108	88	110	90	114
3	Kalyani	25/F	50	88	106	80	98	86	100	89	105
4	Manjula	30/F	55	92	110	85	99	88	105	90	107
5	Murugan	35/M	48	75	99	68	95	74	100	75	100
6	Marimuthamml	40/F	58	82	85	76	80	79	84	80	84
7	Bharathiraja	42/M	60	85	79	78	75	80	78	84	78
8	Vembu	44/M	55	95	82	90	78	92	80	94	81
9	Janaki	40/F	52	92	94	88	89	92	92	94	94
10	Saraswathy	43/F	55	89	82	82	79	86	80	87	84
11	Suresh	42/M	55	90	92	84	88	85	90	88	92
12	Balakrishnan	38/M	50	88	95	80	90	85	92	88	92
13	Jayakumkumar	20/M	50	85	88	78	80	82	85	84	86
14	Subbaram	47/M	52	87	84	80	79	85	82	86	84
15	Radhakrishnan	38/M	54	88	80	80	72	85	78	87	79
16	Marimuthu	32/M	60	92	95	88	89	89	92	90	92
17	Vasantha	30/M	52	90	98	86	90	89	95	89	95
18	Revathi	22/F	50	85	89	78	85	82	88	83	88
19	Thangaraj	38/M	55	78	107	75	100	78	102	80	105
20	Murugesan	40/M	52	80	100	75	92	79	98	82	99

MASTER CHART (MAGNESIUM GROUP)

Sl. No.	Name	Age/ Sex	Wt. (kgs)	Basal		Post induction		Post laryngoscopy		Post intubation (1 min)	
				PR	MAP	PR	MAP	PR	MAP	PR	MAP
1	Sanglimuthu	20/M	55	88	94	78	80	80	84	82	85
2	Natarajan	25/M	58	90	120	82	100	85	104	85	104
3	Balasundar	30/M	52	82	105	72	88	76	91	80	92
4	Kathiresan	35/M	54	86	90	78	64	80	86	81	87
5	Vasantha	40/F	60	82	82	70	69	74	72	75	74
6	Murugeswari	42/F	52	92	90	82	80	84	84	84	84
7	Kamala	18/F	52	88	88	72	83	76	85	76	86
8	Vasunathan	42/M	55	85	106	72	85	74	86	76	89
9	Manoharan	44/M	60	89	97	78	80	80	84	82	87

10	Latha	45/F	52	82	98	75	83	78	89	78	90	
11	Devanai	47/F	55	86	90	78	80	72	84	74	86	
12	Saraswathy	37/F	52	95	110	79	95	82	98	85	99	
13	Kalaiselvi	38/F	54	90	96	75	80	78	82	80	84	
14	Sathiakala	38/F	55	85	90	72	75	72	80	74	82	
15	Suresh	37/M	50	80	82	72	69	74	72	75	74	
16	Jeyakumar	35/M	48	96	88	82	82	84	84	84	86	
17	Mathiventhan	32/M	40	92	97	80	80	83	83	85	85	
18	Srirengam	28/F	50	90	96	75	80	79	82	80	85	
19	Sambanthamoorthy	28/M	50	82	94	71	82	73	84	75	85	
20	Rasiya	25/F	55	85	105	74	85	76	88	76	89	